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DOI: 10.1016/j.emcon.2016.03.006
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Document Version
Publisher's PDF, also known as Version of record

Citation for published version (Harvard):

Link to publication on Research at Birmingham portal

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Hexabromocyclododecane and tetrabromobisphenol-A in indoor dust from France, Kazakhstan and Nigeria: Implications for human exposure

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ABSTRACT

Concentrations of hexabromocyclododecane isomers (α-, β- and γ-HBCDs) and tetrabromobisphenol-A (TBBP-A) were measured — for the first time — in indoor dust from homes, offices and cars from France, Kazakhstan and Nigeria. ΣHBCDs in French and Kazakhstani house dust (median = 1351 and 280 ng g⁻¹, respectively) were consistent with previous reports from the UK and Romania, respectively. Concentrations of ΣHBCDs in Nigerian domestic dust (median = 394 ng g⁻¹) were substantially higher than those reported from Egyptian homes. In general, concentrations of ΣHBCDs in the studied micro-environments were higher than those of TBBP-A, which may be attributed to the major application of TBBP-A as a reactive flame retardant; rendering its release to dust more difficult. Statistical analysis revealed significantly lower ΣHBCDs in French houses than those found in both offices and cars, while ΣHBCDs in cars from Kazakhstan were higher (P < 0.05) than those in homes and offices. Moreover, TBBP-A concentrations in car dust from Nigeria were lower than those found in homes and offices. Exposure estimates revealed higher intake of HBCDs and TBBP-A by toddlers via indoor dust ingestion compared to adults. Combined with their low body weight, this can raise concerns over the potential adverse health effects of such high exposure in toddlers.

1. Introduction

Hexabromocyclododecane (HBCD) and tetrabromobisphenol-A (TBBP-A) are widely used brominated flame retardants (BFRs) with reported global market demands of 170,000 and 31,000 metric tonnes in 2004 and 2011 respectively [1]. HBCD is employed principally as an additive to expanded and extruded polystyrene foams for applications like thermal insulation of buildings, for backcoating of fabrics and to a lesser extent to high-impact polystyrene (HIPS) used in enclosures for electronic equipment. Commercial HBCD formulations consist mainly of the γ-HBCD diastereoisomer (75–89%), while the α- and β-HBCD are present in considerably lower amounts (10–13% and 1–12%), respectively. HBCD has a low water solubility (49, 15, 2 μg L⁻¹ for α-, β-, and γ-HBCD), a fairly low vapour pressure (6.27 × 10⁻² Pa) and is persistent in the environment (estimated t₀.₅ of 51, 1440 and 5760 h in air, water and sediment, respectively). It can therefore bioaccumulate and undergo long-range atmospheric transport [2]. Oral exposure to HBCDs was reported to induce hepatic cytochrome P450 enzymes and alter the normal uptake of neurotransmitters in rat brain. It can
cause disruption of thyroid function, reproductive system, nerve function and development in various classes of vertebrates [3]. Therefore, HBCD was recently included in Annex A of the Stockholm Convention on persistent organic pollutants (POPs) with an exemption for use in expanded polystyrene and extruded polystyrene in buildings [4].

TBBP-A is used mainly as a reactive flame retardant covalently bonded to the polymer matrix in epoxy and polycarbonate resins used in printed circuit boards and electronic equipment. It can also be used as an additive, for instance in HIPS and acrylonitrile–butadiene–styrene resins. Additive usage accounts for ~18% of TBBP-A total applications. However, even when used as a reactive flame retardant, excessive non-polymerized TBBP-A is always present which can be emitted to the environment. Due to its low water solubility (63 μg L⁻¹) and low vapour pressure (6.24 × 10⁻³ Pa), TBBP-A is likely to be associated with suspended particulate matter following release [5]. TBBP-A has been identified as an endocrine disruptor. It also displays a high potency to bind to human transthyretin and was associated with immunotoxic and neurotoxic effects in laboratory animals [6]. The potential toxicity of TBBP-A is mitigated to some extent by its short human half-life (2.2 days) [5]. Therefore, while TBBP-A falls under the REACH registration process due to its high production volume, there are currently no global restrictions on the production and usage of TBBPA or its derivatives. However, the EU risk assessment report on TBBP-A concluded that there is a need for further information and/or testing due to possible degradation to more toxic derivatives (e.g. bisphenol-A) [5].

Several studies have reported on the levels of HBCD isomers and TBBP-A in various biotic and abiotic matrices including air, dust, soil, sediment, human milk, plasma and adipose tissue from various parts of the world, including the Arctic, indicating the ubiquity of these BFRs [6–8]. The few studies available on the levels of HBCDs and TBBP-A in indoor dust have elucidated the significance of inadvertent dust ingestion as a pathway of human exposure to these chemicals, especially for toddlers and young children [9,10]. Moreover, pharmacokinetic modelling of UK adults exposure to HBCDs showed that the levels of HBCDs and TBBP-A in indoor dust samples collected from homes, of an average concentration of 0.5 mg m⁻³, were in the range of adult human exposure to HBCDs and TBBP-A via dust ingestion [9]. This highlights the importance of assessment of human exposure to both HBCDs and TBBP-A via dust ingestion in different indoor microenvironments. Given the reported highly skewed nature of HBCD concentrations in indoor dust from commonly frequented microenvironment categories [9,12], such an assessment would provide a valuable indication of the proportion of the population that may receive elevated exposures as a result of frequenting highly contaminated microenvironments in the course of their daily lives.

Therefore, the current study aims to (a) provide first insights on the levels of HBCDs and TBBP-A in indoor dust samples collected from homes, offices and cars in France, Kazakhstan and Nigeria, (b) compare the dust levels of HBCDs and TBBP-A in the studied countries to those reported from other parts of the world, (c) estimate the daily exposure of adults and toddlers to the target BFRs in the studied countries, and (d) investigate the relative contribution of each microenvironment category to the overall human exposure to HBCDs and TBBP-A via dust ingestion.

2. Materials and methods

2.1. Sampling strategy

All the microenvironments studied comprised a convenience sample of acquaintances of the project team. Dust samples were collected from the following locations: France (Annecy), Kazakhstan (Almaty and Astana) and Nigeria (Lagos). In each country, 3 different microenvironment categories, namely homes (living rooms), offices and cars were sampled. Sampling time and sample numbers are provided in Table 1.

2.2. Sampling methods

Dust samples were collected using a Nilfisk Sprint Plus 1600 W vacuum cleaner or equivalent. Sampling was conducted according to a clearly-defined standard protocol [9] by one of the research team. In offices and homes, one m² of carpet was vacuumed for 2 min and in case of bare floors 4 m² for 4 min. In cars, only the surface of the seats with which occupants would have direct contact (i.e. not including seat backs) was sampled for 2 min. Samples were collected using nylon sample socks (25 μm pore size) that were mounted in the furniture attachment tube of the vacuum cleaner. After sampling, socks were closed with a twist tie, sealed in a plastic bag and stored at −20 °C. Before and after sampling, the furniture attachment was cleaned thoroughly using an isopropanol-impregnated disposable wipe.

3. Analytical protocols

3.1. Sample preparation and extraction

Dust samples were passed through a 500 μm mesh size sieve, weighed accurately and extracted using pressurised liquid extraction ( Dionex ASE-350, Hemel Hempstead, UK). Dust samples (typically between 100 and 300 mg) were loaded into pre-cleaned 66 mL cells containing 1.5 g Florisil and Hydromatrix (Varian Inc., UK) to fill the void volume of the cells, spiked with 20 ng of each of 13C-labelled α-, β-, γ-HBCD and TBBP-A as internal (surrogate) standards (i.e. standards used for determination of analyte concentrations) and extracted with hexane:dichloromethane (1:9, v/v) at 90 °C and 1500 psi. The heating time was 5 min, static time 4 min, purge time 90 s, flush volume 50%, with three static cycles.

3.2. Clean up

The crude extracts were concentrated to 0.5 mL using a Zymark Turbovap® II then cleaned up by loading onto SPE cartridges filled with 8 g of pre-cleaned acidified silica (44% concentrated sulfuric acid, w/w). The analytes were eluted with 25 mL of hexane:dichloromethane (1:1, v/v). The eluate was evaporated to dryness under a gentle stream of N₂, then reconstituted in 100 μL of d₁₅-γ-HBCD (25 pg μL⁻¹ in methanol) as recovery determination (or syringe) standard, used to determine the recoveries of internal standards for QA/QC purposes.

3.3. Analysis

Separation of α-, β-, γ-HBCDs and TBBP-A was achieved using a dual pump Shimadzu LC-20AB Prominence liquid chromatograph equipped with SIL-20A autosampler, a DGU-20A3 vacuum degasser and an Agilent Pursuit XRS3 C₁₈ reversed phase analytical column

<table>
<thead>
<tr>
<th>Country</th>
<th>Location</th>
<th>Time</th>
<th>Number of samples</th>
</tr>
</thead>
<tbody>
<tr>
<td>France</td>
<td>Annecy</td>
<td>Aug–Oct 2008</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>Kazakhstan</td>
<td>Almaty and Astana</td>
<td>May–June 2009</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>Nigeria</td>
<td>Lagos</td>
<td>Sep–Oct 2014</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>10</td>
</tr>
</tbody>
</table>
(150 mm × 2 mm i.d., 3 μm particle size). A mobile phase program based on (a) 1:1 methanol/water and (b) methanol at a flow rate of 150 μL min⁻¹ was applied for elution of the target compounds; starting at 50% (b) then increased linearly to 100% (b) over 4 min, held for 7 min followed by a linear decrease to 60% (b) over 4 min, held for 1 min and finishing with 100% (a) for 10 min. TBBP-A and the three HBCD diastereomers were baseline separated with retention times of 9.0, 10.6, 11.2 and 11.7 min for TBBP-A, α-, β- and γ-HBCD, respectively.

Mass spectrometric analysis was performed using a Sciex API 2000 triple quadrupole mass spectrometer operated in electrospray negative ionization mode. MS/MS detection operated in the MRM mode was used for quantitative determination based on m/z 640.6 → 79, m/z 652.4 → 79 and m/z 657.7 → 79 for the native, 13C-labelled and d13-labelled HBCD diastereomers, respectively and m/z 540.8 → 79, m/z 552.8 → 79 for the native and 13C-labelled TBBP-A, respectively. Specific instrumental calibration parameters are given in Table SI-2.

3.4. Quality assurance/Quality control

Recoveries (average ± standard deviation) of the 13C-labelled internal standards added to the dust samples were: α-HBCD = 82±10%, β-HBCD 85±7%, γ-HBCD 89±12% and TBBP-A 88±9%. No TBBP-A or HBCDs were detected in method blanks (n = 10) consisting of sodium sulfate (0.2 g). Detectable, but very low concentrations of HBCDs (typically 0.1–0.3 ng HBCDs g⁻¹) were obtained for field blanks (n = 7). These consisted of sodium sulfate (0.2 g) “sampled” using the vacuum cleaner and sock according to the standard protocol and treated as a sample. Concentrations in samples in each batch of 10 were thus corrected for the contamination detected in the associated field blank. Method quantitation limits (MQLs) for individual HBCD diastereomers were governed by the field blanks and were typically 0.2 ng g⁻¹, while for TBBP-A the MQLs were 0.15 ng g⁻¹ based on a S/N ratio of 10:1.

The accuracy and precision of the analytical method for HBCDs was assessed via replicate analysis (n = 10) of SRM 2585. The results obtained compared favourably with the indicative values reported elsewhere ([113] and Table SI-4a). For TBBP-A, a standard addition or “matrix spike” method to SRM 2585 at 3 concentration levels (n = 5 at each level) was used to assess the accuracy and precision of the method and good results were obtained (Table SI-4b).

3.5. Statistical analysis

Statistical analysis of the data was conducted using Excel (Microsoft Office 2010) and SPSS version 23.0. In all instances, where concentrations were below the MQL, concentrations were assumed to equal half the MQL. The distribution of each data set was evaluated using both the Kolmogorov-Smirnov test and visual inspection. The results revealed concentrations in all data sets to be log-normally distributed. Hence, ANOVA and t-tests were performed on log-transformed concentrations.

4. Results and discussion

4.1. Concentrations of HBCDs and TBBP-A

The measured levels of HBCDs and TBBP-A in indoor dust showed wide variations between different countries and among microenvironment categories within the same country. A statistical summary of the concentrations of target BFRs in indoor dust is provided in Table 2. Detailed information on the concentrations of α-, β-, γ- HBCDs and TBBP-A in each analysed sample can be found in the supporting information section (Tables SI-1, SI-2 and SI-3). ANOVA revealed significantly higher concentrations (P < 0.05) of ΣHBCDs and TBBP-A in France compared to Kazakhstan and Nigeria. The latter two showed no significant differences in the studied contaminant levels. This may be attributed to the more strict fire-safety regulations in France (as part of the EU) compared to Kazakhstan and Nigeria. Levels of HBCDs in the French dust were generally in line with those reported previously from the UK, USA and China, while it was lower than those reported in domestic dust from other Western European countries including Germany, Belgium and Sweden (Table 3). Concentrations of ΣHBCDs and TBBP-A in house dust from Kazakhstan were also in agreement with those reported in domestic house dust from Romania [14,15], while ΣHBCDs were significantly higher than in domestic dust from the Czech Republic (Table 3). To our knowledge, this is the first report of diastereomer specific HBCD concentrations in indoor dust from Africa and the Middle East. However, the levels of ΣHBCDs in Nigerian dust were significantly higher than ΣHBCD concentrations quantified in domestic dust from Egyptian microenvironments using GC/MS [16]. While the median levels of TBBP-A in dust from Nigerian cars in this study were below MQL, the median TBBP-A concentration in Nigerian houses exceeded those reported in houses studied from Saudi Arabia, Kuwait and Pakistan [15]. In the absence of reliable data on the manufacture, trade and usage patterns of HBCD and TBBP-A, it is difficult to provide an explanation for such variability in BFR levels in indoor dust from different countries. This is further complicated by the general lack of knowledge on the actual sources of HBCDs and TBBP-A in dust from different indoor microenvironments. In this study, we tried to identify potential sources emitting HBCDs and TBBP-A to the indoor dust in our studied microenvironments. We used multiple linear regression analysis to investigate the possible correlation(s) between log transformed BFR concentrations in dust and potential contributing factors (Tables SI-5, SI-6 and SI-7) including the numbers of foam-containing furniture items, TVs, PCs, printers, other electronics (e.g. microwaves, fridges, freezers … etc). For the sampled cars, we examined for correlations with vehicle age, make, country of manufacture and number of electronic devices in the cabin. However, no statistically significant correlations could be established in any of the studied countries. This is consistent with our previous observations in house dust samples from different UK microenvironment categories [9].

4.2. Effect of microenvironment category on BFR levels in dust

Our research group reported previously on significant differences in levels of HBCDs and TBBP-A in indoor dust from different microenvironment categories including homes, offices, cars and public microenvironments in Birmingham, UK [9,17]. To investigate the potential influence of microenvironment category on the levels of target BFRs in dust from France, Kazakhstan and Nigeria, we subjected our data to ANOVA with Games-Howell post hoc testing. Results revealed significantly lower ΣHBCDs in French houses than those found in both of offices and cars. While ΣHBCDs in cars from Kazakhstan were higher (P < 0.05) than those in homes and offices, no significant differences were observed in ΣHBCDs levels in Nigerian dust from different microenvironment categories. For TBBP-A, the only statistically significant difference observed was in Nigeria, where TBBP-A concentrations in car dust were lower than those found in homes and offices. These results are not inconsistent with our previous findings in UK dust where levels of ΣHBCDs in car dust significantly exceeded those in homes and offices while the reverse trend was observed for TBBP-A levels [9]. The high levels of ΣHBCDs in car dust observed in this and previous studies may be attributed to the
application of HBCDs to flame retard car interior fabrics and automotive textiles [12].

The concentrations of TBBP-A in all microenvironment categories in the 3 studied countries were substantially lower than those of HBCDs. This relative order of contamination is not a simple reflection of the respective production volumes of these two BFRs. However, this is consistent with the predominant application of TBBP-A as a reactive BFR, rendering its release from treated goods more difficult than additive BFRs, such as HBCDs and polybrominated diphenyl ethers (PBDEs).

Finally, investigation of the isomer profile of HBCDs in dust from the sampled microenvironment categories (Fig. 1) revealed no statistically significant differences in the studied countries. In general, we observed higher percentage contribution of α-HBCD to ΣHBCDs in indoor dust accompanied by a lower contribution of the γ-isomer than expected from their percentages in the commercial HBCD formulations. This is in agreement with several previous reports of HBCDs in indoor dust (Table 3) and may be attributed to a combination of factors including the interconversion of α- and γ-HBCD at temperatures >160 °C encountered in technical processes required to incorporate HBCD into flame-retarded goods [18]; as well as a photolytically-induced isomerisation in dust that favours the formation of the α-HBCD [19].

4.3. Human exposure to HBCDs and TBBP-A via indoor dust ingestion

The measured concentrations were used to estimate the exposure of adults and toddlers in the studied countries to the target BFRs via indoor dust ingestion. The algorithm given below [43] was used to estimate both adult and toddler exposure to HBCDs and TBBP-A [20].
where $S_{\text{exposure via dust ingestion}}$ is the total daily human exposure to the studied BFRs via dust ingestion (ng day $^{-1}$); $C_{\text{H}}, C_{\text{O}}$ and $C_{\text{C}}$ is the BFR concentration (ng g $^{-1}$) at the respective exposure scenario in homes, offices and cars, respectively. $F_{\text{H}}/F_{\text{O}}/F_{\text{C}}$ is the respective average fraction of time spent in each microenvironment, and $RR$ is the daily dust ingestion rate (mg day $^{-1}$).

We have assumed average adult and toddler dust ingestion figures of 20 and 50 mg day $^{-1}$, and high dust ingestion figures for adults and toddlers of 50 and 200 mg day $^{-1}$ [21]. To our knowledge, there exists no comprehensive dataset that describes time-activity patterns for the population in the studied countries. Hence, our exposure estimates are based on the assumption that dust ingestion occurs pro-rata to typical activity patterns reported previously (i.e. for adults 63.8% home, 22.3% office, 5.1% public microenvironments, 4.1% car, and 4.7% outdoors; for toddlers 86.1% home, 5.1% public microenvironments, 4.1% car, and 4.7% outdoors) [22,23]. However, due to the lack of information on concentrations of our target BFRs in public microenvironments and outdoors in France, Kazakhstan, and Nigeria, for the purposes of our exposure assessment, concentrations of HBCDs and TBBP-A in these microenvironments were assumed identical to those detected in house dust for these countries. We have then estimated various plausible dust ingestion exposure scenarios, the median and 95th percentile concentrations in the dust samples reported in this study (Table 2). It is stressed that the range of exposure estimates via dust ingestion thus derived is only an indication of the likely range within the population. This is due to the highly uncertain nature of the ingestion rates used here (and in other studies) as they are based on a small number of studies involving primary data collection [21,23]. In agreement with previous reports from the UK [9,17], results of this study revealed substantially higher human exposure to $\Sigma$HBCDs than TBBP-A via indoor dust ingestion in France, Kazakhstan and Nigeria (Table 4). In addition, toddlers were more exposed to both BFRs than adults in all the studied countries. This is attributed to a higher dust ingestion rate in toddlers resulting from their increased hand-to-mouth behaviour and lower sense of hygiene. Despite the current lack of internationally-accepted health based limit values for human exposure to HBCDs and TBBP-A, the high intake of these BFRs via dust ingestion in toddlers combined with their low body weight, emphasises the need for further study of the potential adverse health effects of such exposure.

The major fraction of adult exposure to BFRs via indoor dust ingestion occurred at home in both Kazakhstan and Nigeria (Fig. 2). This may be explained by the large fraction of time spent at home.

Table 4

<table>
<thead>
<tr>
<th></th>
<th>$\alpha$-HBCD</th>
<th>$\beta$-HBCD</th>
<th>$\gamma$-HBCD</th>
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<th>TBBP-A</th>
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<td>Adult</td>
<td>Average $^a$</td>
<td>20.8</td>
<td>4.3</td>
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<td></td>
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<td>20.6</td>
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<td>110.7</td>
<td>62.3</td>
<td>100.8</td>
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</table>

$^a$ Estimated using median BFR concentrations in dust and average intake rates of 20 ng day $^{-1}$ and 50 ng day $^{-1}$ for adults and toddlers, respectively.

$^b$ Estimated using 95th percentile BFR concentrations in dust and high intake rates of 50 ng day $^{-1}$ and 200 ng day $^{-1}$ for adults and toddlers, respectively.
However, exposure via ingestion of office dust was predominant in France (Fig. 2). This may be attributed to the significantly higher levels of HBCDs and TBBP-A in French office dust compared to homes (Table 2). Despite the high levels of BFRs in French car dust, the small time fraction spent in this microenvironment category resulted in a minimal contribution to the overall daily exposure via dust ingestion (Fig. 2). Toddlers were mainly exposed to BFRs via ingestion of home dust in all 3 studied countries. However, no child daycare classrooms were sampled in this study. Our research group previously highlighted the significance of classroom dust for exposure of UK toddlers and young children to HBCDs and TBBP-A. Results revealed UK toddlers to be significantly ($P < 0.05$) more exposed to $\Sigma$HBCDs via classroom dust than via house dust [10]. This implies that the exposure of toddlers to $\Sigma$HBCDs in this study may be underestimated.

This study significantly augments the current global database on BFRs by providing the first information on concentrations of HBCDs and TBBP-A in 3 microenvironment categories from France, Nigeria and Kazakhstan. This highlights the ubiquitous nature and global distribution of these flame retardants as indoor contaminants. The higher exposure of toddlers to BFRs via indoor dust ingestion emphasises the need for more studies of the potential adverse health effects of these contaminants as well as for globally integrated actions to reduce and eventually eliminate this potential public health hazard.

Acknowledgements

The authors express their thanks to all the dust donors. Temilola Oluseyi acknowledges gratefully funding from the Commonwealth Scholarship Commission. Collection of the Kazakhstani dust samples was performed by Meruyert Akhanzaripova.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.emcon.2016.03.006.

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